Pericardial Effusion and Giant-cell Arteritis

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L F, aged 55. Housewife

History: Two months of nausea, anorexia and weight loss with upper abdominal pain fifteen minutes after food. Intermittent frontal headaches of constant intensity with night sweats and episodes of palpitations.

On examination: Continuous fever 99–102°F (37·2–38·8°C). Blood pressure 140/80. No abnormal cardiovascular signs. Temporal arteries normally pulsatile and not tender.

Investigations: Hæmoglobin 9·0 g/100 ml, peripheral blood film showed some hypochromia. Serum iron 16 μg/100 ml; iron binding capacity 243 μg/100 ml. Reduced iron stores in bone marrow. Total white count and differential normal. ESR initially 100–140 mm in 1 hour (Westergren).

Protein strip showed low albumin, raised α_1 and α_2 globulins but no paraprotein. Liver function tests normal apart from temporary elevation of alkaline phosphatase and 5′-nucleotidase. Prothrombin time persistently prolonged (20 seconds, control 14 seconds) even after vitamin K_1. Liver biopsy normal.

Culture of blood, urine, sputum, and biopsy material consistently negative (including tubercle bacilli).

Extensive antibody studies in search for infection, autoimmune or collagen disease revealed nothing diagnostic.

Barium follow-through, enema, cholecystogram and intravenous pyelogram all normal. Chest films showed increase in heart size, aortic dilatation, transient small left pleural effusion. Pericardial effusion detected by echocardiogram. Diagnostic tap yielded sterile fluid without malignant cells.

Ultimately temporal artery biopsy revealed giant cells and fragmentation of elastica diagnostic of giant-cell arteritis.

Treatment: Prednisolone 80 mg/day initially. ESR fell from 75 to 15 mm in 1 hour in six days.

Seven weeks after starting prednisolone, a repeat echocardiogram showed virtually no pericardial fluid; reducing dose below 15 mg/day was followed by reaccumulation of fluid and rise in ESR, necessitating increased dosage again.

Recent progress satisfactory.

Comment

Aortic and coronary artery involvement are well described in giant-cell arteritis, but pericardial effusion is a very unusual feature. Hamilton *et al.* (1971) mention it but do not refer to any documented cases, and we have been unable to find any reported examples. Although not susceptible of proof it seems likely that the pericardial effusion in this case is a feature of the giant-cell arteritis, since detailed investigation revealed no other known cause and we have evidence that the effusion resolved following steroid therapy.

Diagnosis was delayed in this patient because of the unusual presentation; headache was not a prominent symptom and the temporal arteries were clinically normal. None of these factors by themselves should delay the taking of a temporal artery biopsy in a patient with anæmia, fever and a high ESR.

REFERENCE
Hamilton C R, Shelley W M & Tumulty P A (1971) *Medicine* **50**, 1

Dr J H Angel (*Watford General Hospital, Watford*) pointed out that the clinical features of giant-cell arteritis, and especially the associated anæmia and high ESR, were very similar to those of the pre-pulseless stage of Takayasu's arteriopathy (*see* Strachan R W, 1964, *Quarterly Journal of Medicine* **33**, 57).

Dr D M Krikler (*Prince of Wales's General Hospital, London N15*) said that giant-cell arteritis responded well to steroids, whereas Takayasu's disease did not; this was a strong point against considering them as one and the same disease.

Dr Angel replied that review of the recent literature (e.g. Soloway M, Moir T W & Linton D S, 1970, *American Journal of Cardiology* **25**, 258) showed that Takayasu's disease often did respond to steroids.

Subphrenic Abscess in a Patient with Crohn's Disease

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A man of 26 presented at Hammersmith Hospital towards the end of 1968 with a history of abdominal distension, severe diarrhœa, a persistent perianal fistula, marked weight loss and multiple joint pains. He was investigated and a diagnosis of Crohn's disease was made. His condition deteriorated and he developed a vesicocolic fistula with pneumaturia. Laparotomy was carried out in January 1969.